

Topic 06 – Hypertension / Vascular disease

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Carotid intima media thickness is related to HIV duration and decreased anti inflammatory status but not to antiretroviral treatment exposure. The CHIC (Collaboration on HIV, inflammation and Cardiova)

Franck Boccara [Orateur] (1), Alexander Boyd (2), Jean Luc Meynard (3), Jacqueline Capeau (4), Assia Samri (5), Ziad Mallat (6), Beny Charbit (7), Pierre Marie Girard (3), Ariel Cohen (1), Moïse Desvarieux (2)
(1) AP-HP, CHU Saint-Antoine, Cardiologie, Paris, France – (2) INSERM 970, Paris, France – (3) AP-HP, CHU Saint-Antoine, Maladies Infectieuses, Paris, France – (4) INSERM UMRS 938, Paris, France – (5) INSERM UMR-S945, Paris, France – (6) INSERM, Paris, France – (7) AP-HP, CHU Pitié-Salpêtrière, Pharmacologie, Paris, France

Purpose: Whether HIV infection itself and/or antiretroviral agents (ARV) have impact on carotid atherosclerosis, assessed using the carotid intima media thickness (IMT), is still debated.

Methods: 100 HIV-infected patients (HIV+) (50 ARV-treated >4 years, 50 ARV-naïve but HIV-infected >2 years) and 50 HIV-negative controls were recruited in age-matched never-smoking male triads. Carotid IMT was measured in a total of 12 segments. Pro- (hs-CRP, Resistin, IL-6, IL-18, Insulin, Serum Amyloid A, D-Dimer) and anti-inflammatory (total and high molecular weight adiponectin, IL-27, IL-10) markers were dichotomized into high/low scores (based on median values). Carotid IMT was compared across HIV/treatment groups, stratified by HIV-infection duration, or inflammatory profiles using linear regression models adjusted for age, diabetes, prior hypertension.

Results: The mean age was 41.2 years (SD=6.7). In HIV+ patients, and median duration of HIV-infection was 7.9 years (IQR=7.6). After excluding 1 elite controller subject (<50 copies/mL, ARV-naïve), and adjusting for nadir CD4+ count, average carotid IMT was thicker with longer (>7.9years) HIV-infection, irrespective of ARV treatment (ARV-treated 760 μ m \pm 10, ARV-naïve 757 \pm 17 μ m) when compared to shorter (<7.9years) HIV-duration (ARV-treated 731 \pm 16 μ m, ARV-naïve 731 \pm 10 μ m). ARV-treated subjects with >7.9 years of HIV-infection had significantly thicker carotid IMT than ARV-naïve subjects with <7.9 years of HIV-infection (P<0.05). Having a low anti-inflammatory profile was associated with thicker carotid IMT, irrespective of high- or low-proinflammatory markers (760 \pm 9 μ m and 768 \pm 13 μ m, respectively) when compared to subjects with high anti-inflammatory profiles (712 \pm 11 μ m and 737 \pm 12 μ m, for high and low pro-inflammatory profile respectively) (p<0.05).

Conclusion: HIV duration, but not ARV treatment, is associated with thicker carotid IMT. Low levels of anti-inflammatory markers highly correlate with thicker carotid IMT.

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Dipping pattern on 24 h ambulatory blood pressure monitoring (ABPM) is related to cardiac infiltration in cardiac amyloidosis

Ludivine Eliahou [Orateur] (1), Vincent Algalarrondo (2), Isabelle Thierry (1), Sylvie Dinanian (1), Denis Chemla (3), Abdeslam Bouzeman (1), Claude Sebag (4), David Adams (5), Michel Slama (1)
(1) AP-HP, CHU Antoine-Béclère, Cardiologie, Clamart, France – (2) AP-HP, CHU Antoine-Béclère, Unité INSERM U 769, Signalisation et Physiopathologie Cardiaque, Cardiologie, Clamart, France – (3) AP-HP, CHU Bicêtre, EA4046 – Réanimation Médicale, Le Kremlin Bicêtre, France – (4) AP-HP, CHU Antoine-Béclère, Clamart, France – (5) AP-HP, CHU Bicêtre, Centre de Référence des Neuropathies Familiales Amyloïdes, Le Kremlin Bicêtre, France

Introduction: Familial amyloid polyneuropathy (FAP) is an autosomic disease caused by the mis-folding of mutated transthyretin. Cardiopathy due

to FAP associates cardiac infiltration and denervation. The aim of the study was to characterize the relationship between blood pressure profile and cardiac amyloidosis.

Methods and results: In a cohort of consecutive 32 FAP patients, we performed ABPM and a standard evaluation including echocardiography, right ventricular catheterism and heart rate variability.

Non-dipping pattern was documented in 81.3%(26/32) in FAP. Non dipper patients displayed higher cardiac infiltration assessed by echo (cf. table). Dipping of systolic blood pressure were negatively correlated to relative wall thickness (RWT) (Rho=-0.038; P=0.047).

Conclusion: A non dipping pattern was common in cardiac amyloidosis due to FAP. This pattern was correlated to the severity of cardiac infiltration measured by echocardiography. ABPM may be a promising tool to evaluate the severity of the myocardial infiltration in cardiac amyloidosis.

Table – Main results (RWT: relative wall thickness)

Variable	FAP total	FAP dipper	FAP non dipper	P
n	32	19% (6)	81% (26)	
age	55,4 \pm 11,3	52,1 \pm 11,3	56,1 \pm 13,3	0.38
% of male	69% (22/32)	83% (05/06)	65% (17/26)	0.71
BMI (kg/m ²)	24,6 \pm 3,9	24,0 \pm 5,8	24,8 \pm 3,6	0.90
echocardiography				
IVS (mm)	12,5 \pm 4,4	10,0 \pm 2,36	13,1 \pm 4,6	0.1
PW (mm)	11,0 \pm 3,0	9,0 \pm 1,2	11,4 \pm 3,2	0.09
LVDD(mm)	44.6 \pm 3.8	45,8 \pm 2,9	44,4 \pm 4.0	0.44
LVEF (%)	60,2 \pm 9.0	65,0 \pm 5,5	59.0 \pm 9,4	0.038
RWT	0,499 \pm 0,153	0,394 \pm 0,055	0,522 \pm 0,158	0.048
Catheterism				
PCWP (mmHg)	8,8 \pm 4,6	6,8 \pm 3,1	9,2 \pm 4,8	0.21
CI (L/min/m ²)	2,7 \pm 0,5	2,9 \pm 0,5	2,7 \pm 0,5	0.35
HRV				
mean heart rate	75,2 \pm 13.9	76,8 \pm 14,1	74.9 \pm 14,2	0.95
sdnn	89.9 \pm 40.7	92,1 \pm 26,5	86,3 \pm 44,2	0.48

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Masked hypertension is frequent in obstructive sleep apnea syndrome and not associated with its severity

Catherine Meuleman [Orateur] (1), Franck Boccara (1), Diane Bodez (1), Louise Boyer-Chatenet (1), Sylvie Lang (1), Ghislaine Dufaitre (1), Stéphane Ederhy (1), Nabila Haddour (1), Xuan-Lan Nguyen (2), Saroumadi Adavane (1), Guillaume Fleury (1), Bernard Fleury (2), Ariel Cohen (1)
(1) AP-HP, CHU Saint-Antoine, Cardiologie, Paris, France – (2) AP-HP, CHU Saint-Antoine, Pneumologie, Paris, France

Background: The prevalence of masked arterial hypertension seems to be high in patients with Obstructive Sleep Apnea Syndrome (OSAS). MH has been associated with a higher risk of cardiovascular events. The aim of this study was to evaluate the prevalence of MH and the different types of arterial hypertension (AH) in a large population of patients with OSAS.

Methods. Clinical blood pressure and twenty-four-hour ambulatory blood pressure monitoring were systematically performed in 253 consecutive patients with documented OSAS. MH was defined as a normal blood pressure (BP) in the clinic or office (<140/90 mmHg), but an elevated BP out of the clinic (ambulatory daytime BP or home BP>135/85 mmHg).

Results. Two hundred and fifty-three subjects (mean age 51.3 \pm 9.5 years, 83% of men, mean BMI 31.8 \pm 5.8 kg/m², mean Apnea-Hypopnea Index (AHI) 52.9 \pm 28.5%) with OSAS were included. Eighty-two patients (32%) had known AH. Among the 171 (68%) without known AH, 55 (32%) were normotensive, 25 (15%) had white coat AH, 52 (30%) had newly diagnosed